

Nutritional Diseases of Poultry

BY HARRY W. TITUS ¹

THIS ARTICLE discusses 16 well-defined nutritional diseases, a group of less-well-defined diseases, and the effect of nutritional deficiencies on growth and reproduction. The material is especially significant because a good deal of rather precise work has been done in this field with poultry.

A FAIRLY LARGE NUMBER of different elements and compounds are required for the normal nutrition of poultry. If one or more of them are not present in the diet in adequate quantity, or if certain ones are present in an unsuitable ratio, there is a disturbance of nutrition, or of the functioning of the body, which may be referred to as a nutritional disease. Other nutritional diseases may result from harmful elements or compounds in the diet.

Knowledge in the field of nutritional diseases of poultry is in a state of active change. That which is believed to be true today may be disproved tomorrow, or, as perhaps more often happens, may prove to be only part of the truth. In several instances an abnormal condition that was originally thought to be the result of a single nutritional deficiency has been found to result from a multiple deficiency. A good example is polyneuritis in the chicken. At one time the cause of this condition was believed to be a deficiency of vitamin B₁, and this vitamin was—and still is—referred to as the antineuritic vitamin and was given the name “aneurin.” It is now known that the condition originally described as polyneuritis gallinarum was produced by diets deficient in vitamins A, E, and G and pantothenic acid as well as in vitamin B₁. Moreover, it has been shown that a deficiency of vitamin B₁ alone does not produce nerve degeneration in the chicken, although it does have other serious effects on the nerves.

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VITAMIN A DEFICIENCY

If day-old chicks are placed on a diet markedly deficient in vitamin A, their rate of growth falls below normal after about 2 weeks and then declines rapidly. The first characteristic symptoms, other than the decrease in rate of growth, are droopiness, a staggering gait, and a ruffled appearance of the feathers. These symptoms may appear as early as the end of the third week. Some of the chicks die before the



FIGURE 1.—Effects of vitamin A deficiency in an advanced stage. Note the swelling around the eye and the cheesy exudate under the lids. (Courtesy of J. R. Beach, Division of Veterinary Science, California Agricultural Experiment Station, Berkeley, Calif.)

end of the fourth week, and most of the others before they are 5 weeks old. Growth usually ceases several days before death occurs. In many of the chicks that survive for more than a week after the first characteristic symptoms appear, the eyes become inflamed and there is a discharge from the nostrils; in some there are a swelling around the eyes and an accumulation of sticky exudate beneath the lids.

With a diet partially deficient in vitamin A, the first symptoms may not appear until the chicks are 5 or 6 weeks old. In this case, a larger proportion of the chicks eventually have inflamed eyes and an accumulation of white cheesy material under the lids (fig. 1).

In mature chickens the symptoms develop much more slowly than in growing chicks, but the inflammation of the eyes becomes strikingly more pronounced. Often there are a white membranous film over the

nictitating membrane, or third eyelid, and a cheesy exudate or discharge, in the conjunctival sacs. There may also be a sticky discharge, either clear or turbid, from the nostrils.

The symptoms of vitamin A deficiency in the turkey poult are in general similar to those in the chick, but according to Hinshaw and Lloyd (21)² the disease is much more acute in poults. These authors described the symptoms in poults that had received little or no vitamin A from the time of hatching as "those of an acute-infectious-contagious disease except that fever was absent." In chicks that were fed the same diet as the poults and kept in the same pen there was a marked nervousness, which the poults did not exhibit.

FINDINGS AFTER DEATH³

An examination of chickens and turkeys that die as a result of vitamin A deficiency reveals lesions, or tissue changes, in many parts of the body, their location

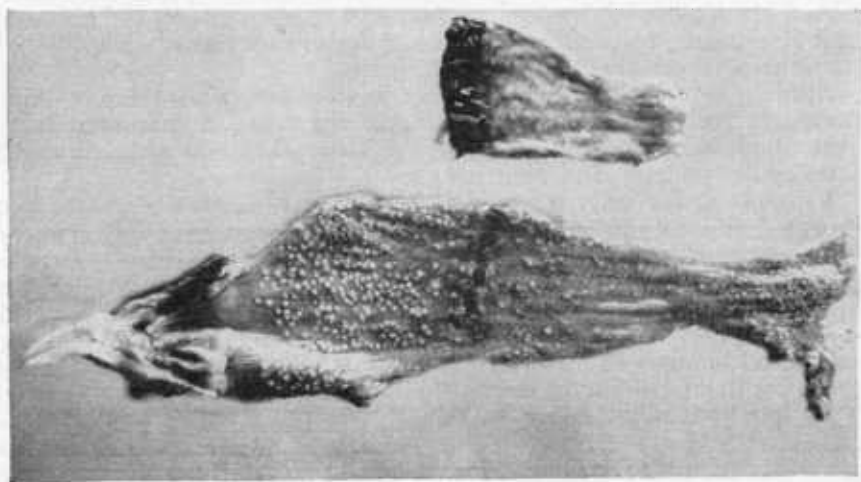


FIGURE 2.—Pustulelike lesions in the esophagus caused by vitamin A deficiency. (Courtesy of L. D. Bushnell, Bacteriology Department, Kansas Agricultural Experiment Station.)

and severity depending to some extent on the age of the bird, the degree of the deficiency, and the length of time between the appearance of the first symptoms and death.

In mature birds lesions resembling pustules are almost invariably found in the mouth, pharynx, and esophagus (fig. 2); in young growing birds these are seen much less frequently. Usually there are white or grayish-white deposits of urates in the kidneys and ureters, which occur more frequently in the chick than in the poult. Sometimes there are deposits of urates on the surface of the heart, liver, and spleen. Hinshaw and Lloyd (21) have reported that they found white, flaky, uratelike deposits between thickened folds of the bursa Fabricii in most of the poults and chicks they examined, and Heywang and Morgan (20) have confirmed their findings in the case of the chick.

² Italic numbers in parentheses refer to Literature Cited, p. 1105.

³ In this article, material on post mortem findings and also material on diseases that seldom or never occur in ordinary poultry production but are of great interest from the experimental standpoint are set in smaller type.

In general, there is a keratinization, or hornlike hardening, of the epithelial cells of the olfactory, respiratory, upper alimentary, and urinary tracts. In severe cases, especially if the birds are mature, virtually every organ in the body may be affected. Also, there are degenerative changes in both the central and peripheral nervous systems, and these explain the staggering gait, which is one of the first symptoms of vitamin A deficiency in chickens, and the extreme lack of muscular coordination in advanced cases.

FUNCTION OF VITAMIN A AND PREVENTION OF DEFICIENCIES

Histological examinations—that is, examinations of the minute structure—of the tissues of chickens that have been fed a diet deficient in vitamin A indicate that one of the functions—if not the primary function—of vitamin A is the proper nourishment and repair of all the epithelial structures, extremal and internal, in the body. In extreme vitamin A deficiency in the chicken the uric acid content of the blood may increase to eight or nine times its normal value. The accumulation of uric acid in the blood and the previously mentioned occurrence of deposits of uric acid in the ureters, the kidneys, and elsewhere are probably results of failure of repair of epithelial structures, especially those of the kidneys.

Studies made with other animals have shown that vitamin A is necessary for the normal functioning of the eyes. Apparently, however, it plays no detectable role in the absorption and metabolism of fats, carbohydrates, and proteins.

Vitamin A has been referred to as the anti-infective vitamin, but repeated attempts to show that it affects the mechanisms that give the body immunity against infections have failed. When the diet is deficient in vitamin A, however, the epithelium, or surface layer, of the mucous membranes is damaged, and as a result the entry of bacteria is made easier. Thus, although vitamin A is of no value in making an animal immune to infectious diseases, it is of value in maintaining the "first line of defense," the epithelial structures.

As has been pointed out by Barger and Card (2), a partial deficiency of vitamin A in diets for poultry is more common than is generally supposed. They state that it is especially likely to occur in regions where the summers are hot and dry and there is a resulting shortage of green forage. Partial vitamin A deficiency is often an aftermath of drought. A partial vitamin A deficiency is also possible when flocks are closely confined unless an adequate supply is included in the feed.

The obvious method of preventing the development of vitamin-A-deficiency disease in poultry is to supply an adequate quantity of this vitamin in the feed. The minimum vitamin A requirement of the growing chick is about 675 to 775 International units per pound of feed; that of the growing poult is about $2\frac{1}{2}$ times as much. An adequate supply, as distinguished from the minimum, is about 1,450 International units per pound of feed for the chick and about 3,650 per pound of feed for the poult. The vitamin A in mixed feeds, however, is not very stable, and for this reason it is a good practice to formulate the diets of chicks and poults so that they will contain 3,000 and 7,500 International units per pound of feed, respectively. The feed of chickens kept for egg production should contain approximately 3,150

International units per pound. The feed of breeding stock—chickens or turkeys—should contain about 4,720 units per pound.

The approximate vitamin A contents of some of the richer sources of this vitamin used in feeding poultry are as follows:

	<i>International units per pound</i>
Fortified cod-liver oil.....	1,360,770
Fortified sardine oil.....	1,360,770
Cod-liver oil.....	385,550
Sardine oil.....	52,000
Alfalfa-leaf meal, dehydrated.....	95,000
Alfalfa-leaf meal.....	32,000
Alfalfa meal.....	13,000
Corn-gluten meal.....	6,800
Yellow corn.....	3,180

VITAMIN B₁ DEFICIENCY

The symptoms of vitamin B₁ deficiency are similar in chickens and turkeys and are essentially the same at different ages. A diet containing little or no vitamin B₁ but otherwise adequate causes a prompt decrease in appetite, followed soon by a steady decline in live weight. After 7 to 10 days there is progressive development of general paralysis. The extensor muscles of the legs are the first

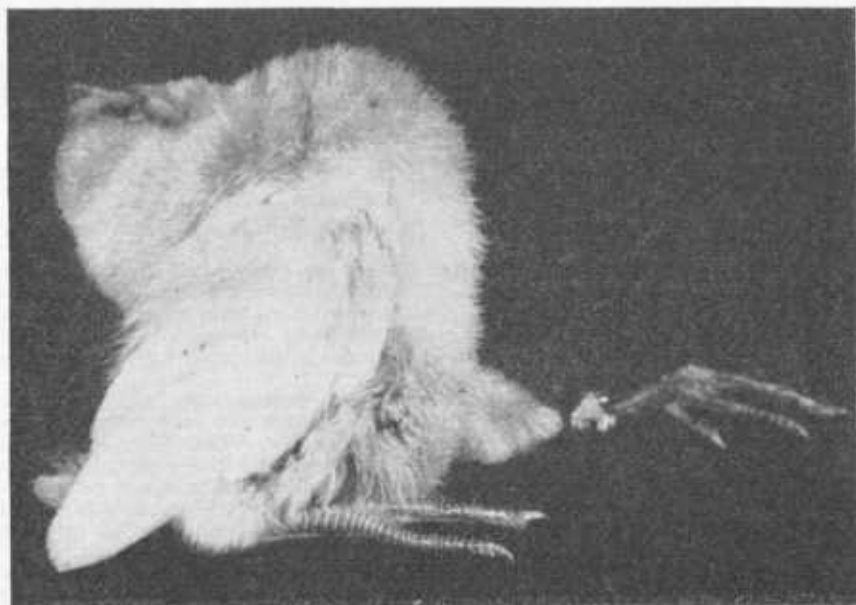


FIGURE 3.—Head retraction is a characteristic symptom of vitamin B₁ deficiency in the late stage. (Courtesy of L. C. Norris, Nutrition Division, Department of Poultry Husbandry, Cornell University.)

to become affected, but soon the paralysis extends to the wings and neck and, finally, to all the muscles. In the early stages of paralysis the bird swallows feed or water with great difficulty; in the later stages the body temperature falls, and the head is raised and drawn back. Death follows, usually within 1 or 2 days after the typical symptom of head retraction appears (fig. 3).

When the diet is only partially deficient in vitamin B₁, 30 days or more may elapse before the symptoms of paralysis appear. In the ability of chickens and other birds to survive on diets that are only partially deficient in vitamin B₁, there are marked individual differences. In adult and nearly grown birds there is a loss of about 20 percent of the initial live weight before death occurs.

The first symptom in pigeons is a partial loss of the ability to walk. The next symptom is uncontrolled movement of the head. Finally, head retraction occurs, and the legs are drawn up close to the body; often the bird turns somersaults. As in the chicken, death follows soon after head retraction occurs.

FINDINGS AFTER DEATH

With few exceptions, the diets that have been used in studying vitamin B₁ deficiency have not contained adequate quantities of some of the other vitamins, especially vitamin A and those that commonly occur with vitamin B₁ in natural products. As a matter of fact, the diets that were used in the first studies of vitamin B₁ deficiency in the chicken and pigeon were also markedly deficient in vitamin A. Many of the earlier descriptions of the findings after death from what was considered to be vitamin B₁ deficiency were for this reason in reality descriptions of the results of a multiple deficiency.

For many years it was believed that a deficiency of vitamin B₁ caused extensive degeneration of the peripheral nervous system. Engel and Phillips (11) have shown, however, that there is no nerve degeneration in either the rat or the chick when the vitamin-B₁-deficient diet contains a fully adequate supply of vitamin A and vitamin G. Moreover, there is evidence that the withholding of feed produces in the chicken and other animals many of the changes that are observed when a diet deficient in vitamin B₁ is fed.

At the time this is being written there is not available to the writer a good description of the post-mortem findings in birds that have died as a result of an uncomplicated vitamin B₁ deficiency.

FUNCTION OF VITAMIN B₁ AND PREVENTION OF DEFICIENCIES

According to the evidence now available, vitamin B₁ is required for the proper metabolism of carbohydrates.⁴

Nitzescu and Ioanid (39) found that if hens are deprived of vitamin B₁ the sugar content of their blood decreases and remains below normal for 10 to 14 days, then rises rapidly in the next few days to more than twice normal. If vitamin B₁ is injected, the sugar content of the blood returns to normal. They found also that injections of vitamin B₁ produce appreciable decreases in the sugar content of the blood of normal hens.

The animal has relatively little capacity for storing vitamin B₁, and for this reason, when there is a multiple deficiency of this and other vitamins, the symptoms of vitamin B₁ deficiency tend to appear first. That these symptoms, paralysis and head retraction—the first except cessation of growth—are the result of vitamin B₁ deficiency may be shown conclusively by causing them to disappear in a short time—often less than 2 hours—by the administration of synthetic crystalline vitamin B₁. That these symptoms can thus be made to disappear is evidence that they are not the result of nerve degeneration.

Vitamin B₁ deficiency in poultry is rarely observed under practical conditions. It can be produced by feeding a diet that consists wholly of polished rice or degerminated grain or specially formulated diets.

In practical poultry production no special precautions need be taken to prevent vitamin B₁ deficiency. The minimum vitamin B₁ requirement of poultry is about 90 to 135 International units per pound of feed. An adequate supply is about 180 International units per pound of feed. Most diets for poultry contain two to three times this quantity.

⁴ More specifically, soon after vitamin B₁ is absorbed from the intestinal tract, it is converted into co-carboxylase, which functions as a coenzyme in the metabolism of pyruvic acid. When the diet does not contain enough vitamin B₁, pyruvic acid accumulates in various tissues of the body and exerts a toxic effect on the nervous system.

The approximate vitamin B₁ contents of some of its richer sources in feeding poultry are as follows:

	<i>International units per pound</i>
Yeast, brewers', dried.....	4,500
Soybean meal.....	1,600
Oats.....	1,200
Wheat middlings, standard.....	1,000
Wheat bran.....	840
Wheat.....	680
Skim milk, dried.....	500
Buttermilk, dried.....	450
Barley.....	400
Corn.....	.400

VITAMIN B₆ DEFICIENCY

Very little is known about the vitamin B₆ requirements of poultry or the symptoms of vitamin B₆ deficiency. In 1939 Jukes (23) reported that the symptoms in chicks consist of slow growth, depressed appetite, and inefficient utilization of feed, followed in some cases by spasmodic convulsions and death, but in 1940 he reported (24) that the diet that he had used in studying vitamin B₆ deficiency was deficient even when supplemented with the vitamin. More recently, Hegsted, Oleson, Elvehjem, and Hart (19) reported that the only symptoms of vitamin B₆ deficiency that they had observed in growing chicks were lack of growth and extreme weakness.

Vitamin B₆ is widely distributed in nature, and for this reason a deficiency does not occur among poultry that are fed practical diets but would be produced only by special, highly simplified diets. Yeast, wheat germ, and egg yolk are some of the best sources of B₆ vitamin; other good sources are kidney, liver, fish meal, dried skim milk, dried buttermilk, alfalfa products, and rice polishings; all the grains appear to be fairly good sources.

VITAMIN D DEFICIENCY AND RICKETS

Abnormal development of the bones of growing chickens, turkeys, and other kinds of poultry may result from a number of dietary causes, among which are (1) a deficiency of one or more of the following substances: Vitamin D, calcium, phosphorus, manganese, and choline; (2) a marked unbalance of calcium and phosphorus; and (3) the presence of certain substances that make the vitamin D or the phosphorus unavailable. The discussion here is restricted to those conditions in which the absorption and metabolism of vitamin D, calcium, and phosphorus are directly involved. The condition that results from a deficiency of manganese or choline or both is discussed later.

Some writers—for example, McGowan and Emslie (34)—distinguish between rickets and another condition of the bones, osteoporosis, on the ground that the former is the result of a deficiency of phosphorus and the latter of a deficiency of calcium. The distinction is warranted because the changes that occur in the bones are not the same in the two conditions. However, both may be prevented or cured by including sufficient vitamin D in the diet, unless the deficiency of phosphorus or calcium is very marked.

At the time of hatching, the chick is essentially osteoporotic, that is, its bones have a much lower calcium-phosphorus ratio than they do later on; hence it requires an immediate supply of calcium in

its diet. If the diet is deficient in calcium or if that which is present is unavailable as a result of a deficiency of vitamin D, the osteoporotic condition becomes more pronounced. If there is an adequate supply of calcium but a deficiency of phosphorus or vitamin D or both, rickets develops.

Rickets may be produced on diets that contain adequate quantities of vitamin D, calcium, and phosphorus if the diets also contain large quantities of certain inorganic compounds, such as soluble salts of iron, lead, and beryllium. This is because iron, lead, and beryllium form insoluble compounds with the phosphorus and make it unavailable. Excessive quantities of calcium in the diet of growing chicks may also make much of the phosphorus unavailable as a result of the formation of the relatively insoluble calcium phosphate. A diet deficient in vitamin D is therefore more rachitogenic (rickets-causing) if it contains an excessive quantity of calcium than if it contains a much smaller but adequate quantity.

A condition called sulfur rickets may result from the inclusion in the diet of 2 percent or more of sulfur for the control of coccidiosis. If the particles of sulfur are very small (for example, colloidal), less than 2 percent may cause this condition. Why sulfur has this effect is not known, but there are reasons for believing that it interferes with the absorption of vitamin D. The condition is relieved but not entirely eliminated by doubling or trebling the vitamin D content of the diet. Sunshine appears to be more effective than vitamin D in preventing sulfur rickets.

The rickety and osteoporotic conditions encountered in the practical production of poultry are most frequently caused by a deficiency of vitamin D rather than by a deficiency of calcium or phosphorus or the presence in the diet of large quantities of soluble salts of iron, beryllium, or lead.

If a vitamin-D-deficient diet is fed, beginning with the first feeding, the first symptoms usually make their appearance in the poult toward the end of the third week and in the chick about a week later. Usually most of the poults die within 5 weeks and a large proportion of the chicks within 8 weeks.

In the adult chicken the first symptom of vitamin D deficiency is a thinning of the shells of its eggs. If the deficiency is marked, there is a fairly prompt decrease in both egg production and hatchability. After a time the breast bones become distinctly less rigid. Adult chickens, however, can live for months on a diet that supplies practically no vitamin D.

The first symptoms of vitamin D deficiency in the growing chicken and turkey are a tendency to rest frequently in a squatting position, a disinclination to walk, and a lame, stiff-legged gait. These symptoms are readily distinguishable from those of vitamin A deficiency in that in vitamin D deficiency the chick or poult at first is alert rather than droopy and walks with a lame rather than a staggering gait. Other symptoms, in the usual order of their occurrence, are retardation of growth, enlargement of the hock joints, beading at the ends of the ribs, and marked softening of the beak (fig. 4). As in many other



FIGURE 4.—Vitamin D deficiency in a young chicken. Note the crossed beak, which is soft and rubbery, and the inability to stand.

nutritional diseases of poultry, the feathers soon acquire a ruffled appearance.

GROSS CHANGES AND CHEMICAL FINDINGS

In the chick and poult, vitamin D deficiency produces marked changes in the bones and the parathyroid and thyroid glands and variable changes in the calcium and phosphorus content of the blood. The bones may be soft or only moderately so, but in any case their ash (mineral) content is much less than normal, and in some instances the ash content of the tibia (drumstick bones) may be as little as 27 percent on a moisture-and-fat-free basis. (The normal ash value for the tibiae of young chicks is about 46 percent.) The epiphyses, or growing ends, of the long bones are usually enlarged. The parathyroid becomes enlarged, sometimes to eight times its normal size, as a result of an increase in both the size of the cells and the number of epithelial cells. At most there is no great change in the size of the thyroid, but there is an appreciable increase in the number of cells.

The changes in the calcium and phosphorus content of the blood depend on the calcium and phosphorus content of the diet. If the diet has a high calcium content, the calcium content of the blood may be approximately normal and the phosphorus content low. In such a case the bones may be somewhat rarefied rather than soft. If there is a deficiency of both calcium and phosphorus in the diet, the blood may contain less than the normal quantities of these elements. When there is a deficiency of phosphorus, the bones tend to be soft and may be bent.

In adult chickens a deficiency of vitamin D eventually produces changes in the parathyroid similar to those produced in chicks. The bones tend to become rarefied (osteoporotic) rather than soft.

FUNCTION OF VITAMIN D AND PREVENTION OF DEFICIENCIES

It must be concluded that vitamin D is required for the normal metabolism of calcium and phosphorus in the chicken, but the exact manner in which it performs its function is not known. A diet deficient in vitamin D does not produce rickets in rats if it contains suitable quantities of calcium and phosphorus, but rickets is always produced in chickens by a deficiency of vitamin D, even when the diet contains calcium and phosphorus in suitable quantities. There is good evidence that in the rat vitamin D regulates the absorption of calcium and phosphorus from the intestine, and it is highly probable that it performs the same function in the chicken.

Before the discovery of the importance of vitamin D in the nutrition of poultry, it was not possible to raise poultry in strict confinement; that is, without access to sunshine. Even under normal conditions, rickets was likely to occur in poultry whenever there were long periods of cloudy or rainy weather during the brooding season. It is now a common practice to include at least some vitamin D in the diet of poultry whether they have access to sunshine or not. Even so, rickets is occasionally encountered as a result of using an inferior grade of cod-liver oil or other source of vitamin D.

Laying flocks are frequently housed in quarters into which little or no sunshine penetrates, and many such flocks suffer from the effects of vitamin D deficiency. Even when laying flocks are allowed to range the year round, they may get too little sunshine during the late fall, winter, and early spring. It is advisable, therefore, to include some vitamin D in the diet of all laying stock, whether or not they are allowed to range.

The usual sources of vitamin D, other than sunshine, are cod-liver oil, sardine oil, certain other fish oils, and "D"-activated animal sterol. Vitamin D deficiency is readily prevented or cured by a suitable quantity of any one of those materials. The minimum vitamin D requirement of the growing chick is about 60 to 90 A. O. A. C. chick units⁵ per pound of feed; that of the growing poult is about 2 to 3½ times as much. An adequate supply for the chick is about 180 A. O. A. C. chick units per pound of feed and for the poult about 360 units. The duckling apparently requires about as much vitamin D as the poult.

Chickens that are being kept for the eggs they produce should receive about 360 A. O. A. C. chick units of vitamin D per pound of feed, and breeding stock—both chickens and turkeys—about 540 units.

Cod-liver oil that is sold as such in interstate commerce is required by law to contain not less than 85 United States Pharmacopoeia units of vitamin D per gram, or about 38,560 U. S. P. units per pound. In the case of cod-liver oil 1 U. S. P. unit of vitamin D is equal to 1 A. O. A. C. chick unit. The fortified cod-liver oil and sardine oils now on the market are usually guaranteed to contain about 181,600 A. O. A. C. chick units per pound. Other oils and other products used as sources of vitamin D in feeding poultry are usually

⁵ The standard unit of the Association of Official Agricultural Chemists.

sold on the basis of a guaranteed number of A. O. A. C. chick units per gram or per pound.

VITAMIN E DEFICIENCY

By feeding a diet high in fat but markedly deficient in vitamin E to chicks, ducklings, and poults, Pappenheimer, Goettsch, and Jungherr (41) produced nutritional encephalomalacia (crazy-chick disease) in the chicks, nutritional myopathy (a disease of the muscles) in the ducklings, and nutritional myopathy of the gizzard in the poults. They were able to prevent or at least greatly reduce the incidence of the encephalomalacia in the chicks by including various vegetable oils in their diet. They completely prevented the development of encephalomalacia by administering small quantities of alpha-tocopherol (vitamin E) by mouth. In a single experiment with ducklings the nutritional myopathy was prevented by substituting 5 percent of hydrogenated cottonseed oil for an equal weight of lard in the diet. In the case of poults the administration by mouth of 0.34 cubic centimeter of wheat-germ oil per head per day in gelatin capsules greatly reduced the incidence of the gizzard condition but did not eliminate it completely.

SYMPTOMS OF NUTRITIONAL ENCEPHALOMALACIA IN THE CHICK

When a diet such as that used by Pappenheimer and associates (41) is fed to day-old chicks, the encephalomalacia may occur as early as the seventh and as late as the fifty-sixth day, but the highest incidence is between the fifteenth and thirtieth days after hatching. The average age at which the disease occurs is about 5 weeks. In older chicks the average number of days before the onset of the disease depends on the age at which the deficient diet is first fed; the older the chicks are, up to 8 weeks of age, the more quickly they are affected. The disease rarely occurs among chicks more than 8 weeks old.

The symptoms of nutritional encephalomalacia are described very well by its popular name, "crazy-chick disease." When the chicks attempt to walk, they often fall forward, backward, or to one side and then wheel in circles. In advanced cases there is frequently complete prostration, with the legs extended, the head sometimes retracted, and tremors of both head and legs (fig. 5).



FIGURE 5.—A chicken prostrated by nutritional encephalomalacia, or crazy-chick disease, in an advanced stage. The retracted head is also a result of vitamin E deficiency. (Courtesy of Erwin Jungherr, Department of Animal Disease, Storrs Agricultural Experiment Station, Storrs, Conn.)

Extensive lesions are usually found in the brains of chicks that have died of nutritional encephalomalacia. The cerebellum (the hind part of the brain) is most commonly affected; but in somewhat more than 25 percent of all cases lesions are found also in the cerebral hemispheres, and in about 12 percent of all cases in the medulla. In some cases four-fifths of the cerebellum may be affected, and in others the lesions may be so small that they cannot be detected with the unaided eye. The affected tissues change from pink to greenish yellow and in the healing stage to a rusty brown. For a rather complete description of the microscopic changes that take place in the brain, the reader is referred to Pappenheimer, Goettsch, and Jungherr's monograph (41).

SYMPTOMS OF NUTRITIONAL MYOPATHY, OR MUSCLE DISEASE

The symptoms of nutritional myopathy in ducklings appear quite suddenly, usually in the second or third week. In the early stages the ducklings walk awkwardly, with their feet turned in and sometimes overlapping. Often they are found sprawled out and unable to rise. Sometimes there are coarse tremors. Only the skeletal muscles show pathologic changes; the muscles are pale in color—a light creamy yellow rather than dark red. There are widespread hyaline necrosis and some edema, or watery swelling.

When the Pappenheimer-Goettsch diet is fed to young poults, there are no specific outward indications that anything is wrong, but on post mortem examination, lesions or tissue changes are found in the muscular wall of the gizzard. Histologically, the changes in the muscles of the gizzard are hyaline necrosis and fibrosis.

OTHER CONDITIONS ATTRIBUTABLE TO A DEFICIENCY OF VITAMIN E

By feeding special diets to chicks, Dam and Glavind (9) produced a condition, which they called alimentary (nutritional) exudative diathesis, that could be cured by adding synthetic vitamin E (d,l alpha-tocopherol) to the diets. The condition was characterized by an accumulation of large quantities of transparent fluid in the subcutaneous tissues. The accumulations were found in various parts of the body but most frequently in the breast and abdomen. The fluid had the same composition as blood plasma and clotted readily. In addition to the accumulations of fluid, hyperemia (excess of blood), slight hemorrhage, and accumulation of white blood corpuscles in connective tissues were observed.

Bird and Culton (3) have described a generalized edema which they produced in young chicks by feeding a diet of dried skim milk, dextrinized cornstarch, cod-liver oil, and mineral salts. This diet is deficient in vitamin E and in other nutritional factors, but Bird and Culton were able to prevent the development of the edema by administering d,l alpha-tocopherol.

Hammond, at the Beltsville Research Center, Beltsville, Md., produced crazy-chick disease by feeding diets that contained 3 percent or more of cod-liver oil to day-old chicks. Mild cases were cured within a few days by administering synthetic vitamin E. This condition was essentially the same as the crazy-chick disease some-

times observed in commercial flocks, and the brain lesions were like those found by Pappenheimer, Goeltsch, and Jungherr (41) in nutritional encephalomalacia.

OCCURRENCE AND PREVENTION OF VITAMIN E DEFICIENCY

Crazy-chick disease, or nutritional encephalomalacia, occurs occasionally in commercial flocks that are fed typical feed mixtures for poultry. In such cases it has been often found that the feed mixture was prepared several months before it was used, strongly suggesting that the vitamin E originally in the feed was destroyed or inactivated before the feed was used.

Much can be done to avoid the destruction or inactivation of the vitamin E in feed mixtures by not using excessive quantities of cod-liver oil or other fats and oils and by feeding all mixtures within a short time after they are prepared.

Very few quantitative data are available on the vitamin E content of feedstuffs, and little is known about the quantitative requirements of poultry for vitamin E. It is known, however, that good sources of vitamin E include wheat-germ meal, alfalfa, alfalfa meal, alfalfa-leaf meal, wheat middlings, wheat shorts, wheat bran, and all unground grains and seeds.

When nutritional encephalomalacia occurs in a flock, it can be checked, and the individual cases that do not become acute can be cured, by adding to the diet 1 to 2 percent of corn oil, soybean oil, peanut oil, wheat-germ oil, or cottonseed oil for a few weeks. Experience has shown that the addition to the diet of more than 2 percent of such oils is often much less effective than the addition of 1 percent.

VITAMIN G DEFICIENCY

The characteristic symptom of vitamin G deficiency in the chick is a condition referred to as curled-toe paralysis, but, according to Norris, Wilgus, Ringrose, and others (40) and Stokstad and Manning (48), this condition does not occur if the diet is so extremely deficient in vitamin G that the chick dies. If a small quantity of vitamin G is added to an extremely deficient diet, the paralysis occurs, while if a sufficiently large quantity is added the paralysis is prevented. Three degrees of severity of curled-toe paralysis in chicks have been described by Stokstad and Manning (48). The first degree is characterized by a tendency to rest on the hocks and a slight curling of the toes, the second by marked weakness of the legs and a distinct curling of the toes of one or both feet, and the third by toes that are completely curled inward or under and a weakened condition of the legs that compels the chicks to walk on their hocks.

Other symptoms of vitamin G deficiency in the chick are a marked decrease in the rate of growth or even complete failure to grow, diarrhea after 8 or 10 days, and a high mortality rate after about 3 weeks. According to Lepkovsky and Jukes (31) the growth of the feathers appears not to be impaired. These workers have reported that, as a matter of fact, the main wing feathers appear to become disproportionately long.

The symptoms of vitamin G deficiency in the poult were found by Lepkovsky and Jukes (31) to be different from those in the chick. According to these workers, a dermatitis, or skin inflammation (fig. 6), appears in young poult after about 8 days, and the vent becomes

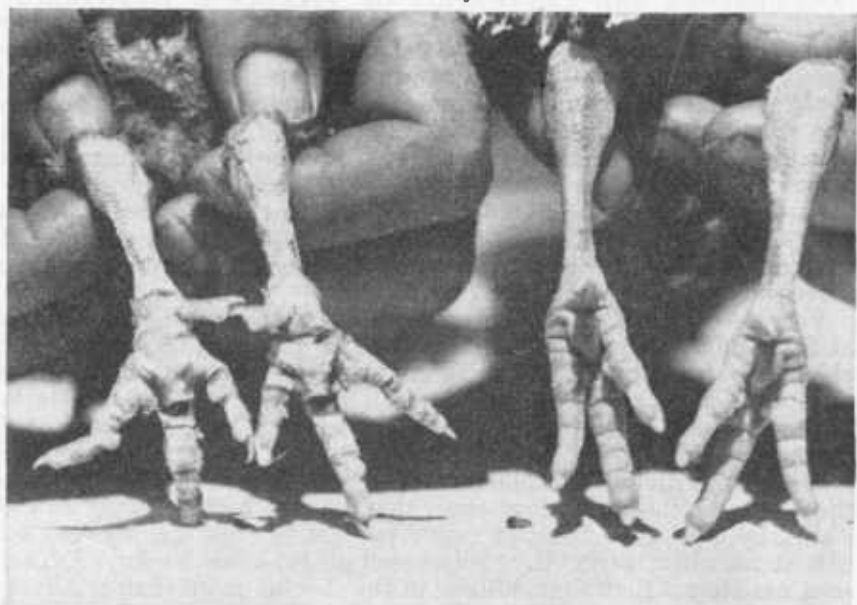


FIGURE 6.—Vitamin G deficiency in a turkey poult. Note the swelling, scaling, and fissuring in the feet of the poult at the left, produced by feeding a diet deficient in vitamin G. The poult whose normal feet are shown at the right received the same diet with the addition of a vitamin G concentrate made from whey. (Courtesy of Thomas H. Jukes, Division of Poultry Husbandry, California Agricultural Experiment Station, Davis, Calif.)

encrusted, inflamed, and excoriated, or stripped of skin. Growth slows up and ceases completely by about the seventeenth day, and deaths begin to occur about the twenty-first day.

FINDINGS AFTER DEATH

According to Phillips and Engel (42) a deficiency of vitamin G in the diet of the chick produces specific changes in the main peripheral nerve trunks. In acute cases there are hypertrophy (increase in cell size) of the nerve trunks and a readily observable change in their appearance. Degenerative changes also appear in the myelin of the nerves. Phillips and Engel also found congestion and premature atrophy (wasting) of the lobes of the thymus. The kidney, thyroid and suprarenal glands, brain, and brain stem appeared not to be affected.

FUNCTION OF VITAMIN G AND PREVENTION OF DEFICIENCIES

It is known that vitamin G is an essential component of certain enzyme systems and that it has some functions in the oxidation proce-

esses of the cell. Just what happens when these enzyme systems fail is not definitely known, but the evidence available indicates that the growing chick requires vitamin G for the normal functioning and maintenance of the nervous system, particularly the main peripheral nerve trunks.

Relatively few of the feedstuffs used for poultry contain enough vitamin G to meet the minimum requirement of the chick or poult during the first few weeks of life; hence if the ingredients of the diet of the young chick or poult are not selected so as to include one or more of the richer sources of vitamin G, the diet is likely to be deficient in this vitamin. In the case of chicks and poults that are closely confined this is especially true unless green feeds or other good sources of vitamin G, such as dried skim milk, dried buttermilk, and alfalfa-leaf meal are supplied.

The minimum vitamin G requirement of the growing chick and poult varies with their age. During the first week it is about 1,300 micrograms per pound of feed for the chick and about 1,600 micrograms for the poult. The duckling's requirement is about the same as that of the chick. An adequate supply of vitamin G for the first 4 or 5 weeks for all three species is about 1,670 micrograms per pound of feed.

The vitamin G requirement of the adult chicken, turkey, or duck is relatively low, but it increases somewhat with the onset of egg production. Chickens being kept for the eggs they produce probably require only 600 to 800 micrograms of vitamin G per pound of feed, but the feed of breeding stock—chickens or turkeys—should contain about 1,250 micrograms per pound to maintain a high hatchability.

The approximate vitamin G contents of some of the richer sources of this vitamin used in feeding poultry are as follows:

	<i>Micrograms per pound</i>
Yeast, brewers', dried.....	16,000
Dried buttermilk (sweet cream).....	12,000
Dried whey.....	10,000
Dried buttermilk.....	9,000
Dried skim milk.....	9,000
Alfalfa-leaf meal, dehydrated.....	8,000
Alfalfa-leaf meal.....	7,000
Alfalfa meal.....	5,000
Fish meal, whitefish.....	4,200
Fish meal, sardine.....	3,200
Meat scrap.....	3,000
Soybean meal.....	1,500

VITAMIN K DEFICIENCY

Apparently vitamin K is necessary for the formation of prothrombin, which in turn is necessary for the normal clotting of blood.

If very young chicks are fed a diet deficient in vitamin K, the time required for their blood to clot begins to increase after 5 to 10 days and becomes greatly increased after 7 to 12 days. After about a week on such a diet, hemorrhages often occur in any part of the body, spontaneously or as the result of an injury or bruise. The only external symptoms of vitamin K deficiency are the resulting accumulations of blood under the skin.

Chicks on a vitamin-K-deficient diet become anemic after a time as a result of the hemorrhages. Examination after death often reveals accumulations of

blood in various parts of the body, and there are invariably erosions of the gizzard lining (1).

The symptoms of vitamin K deficiency may be produced quite easily in young chicks in the laboratory, but they are seldom if ever observed when the chicks are raised in the usual manner. The age at which the vitamin-K-deficient diet is first fed influences the development of the resulting deficiency disease. The younger the chicks, the more susceptible they are; the deficient diet does not cause the disease after the chicks are a few weeks old. Hemorrhages may be produced within 12 to 20 days in adult chickens, however, by tying off the bile ducts, indicating that bile is necessary for the absorption of vitamin K.

Vitamin K has been found in such diverse materials as dried alfalfa, fish meal, and rice bran that had been moistened and allowed to stand at room temperature for a few days, kale, tomatoes, hempseed meal, and hog-liver fat. In corn, wheat, or rice there appears to be little or no vitamin K. In any case, it may be pointed out that in compounding practical diets for poultry it is not necessary to take special precautions to insure an adequate supply of vitamin K.

PANTOTHENIC ACID DEFICIENCY ⁶

SYMPTOMS

The symptoms of pantothenic acid deficiency in the chick, according to Ringrose, Norris, and Heuser (46), are as follows: Growth is retarded, and the feathers become ragged in appearance. Within 12 to 14 days the margins of the eyelids become granulated, and frequently a viscous exudate, which causes the eyelids to stick firmly together, is formed. Crusty scabs appear at the corners of the mouth (fig. 7), and the skin on the bottoms of the feet often becomes thickened and cornified. At first there is no loss of down or feathers, but after about 18 weeks complete loss of feathers in limited areas on the head and neck may occur.



FIGURE 7.—Pantothenic-acid deficiency. Note the lesions at the corner of the mouth and on the eyelids, which are stuck together.

The symptoms of pantothenic acid deficiency and egg-white injury (see the next section) are very similar, but according to Jukes ⁷ the two conditions may be distinguished from

⁶ The nutritional factor now called pantothenic acid has been referred to at various times as the chick antipellagra factor, the filtrate factor, the chick antidermatitis or antidermatosis factor, and the antidermatosis vitamin.

⁷ Personal communication from T. H. Jukes, University of California, Davis, Calif.

each other as follows: In egg-white injury the first symptom is a roughening of the skin below the lower mandible, whereas the dermatitis, or inflammation of the skin, observed in pantothenic acid deficiency appears first at the corners of the mouth and is seldom seen below the mandible. In egg-white injury the feet become involved at the same time as the mouth, whereas dermatitis of the feet is rarely seen in pantothenic acid deficiency and then only in the later stages—usually about 2 or 3 weeks after dermatitis has appeared at the corners of the mouth.

The characteristic dermatitis produced in chicks by feeding diets deficient in pantothenic acid has not been found in adult chickens fed similar diets.

FINDINGS AFTER DEATH

Ringrose, Norris, and Heuser (46) reported that on post mortem examination of the affected chicks a puslike substance was frequently observed in the mouth and an opaque, grayish-white exudate in the proventriculus. The entire intestinal tract was found to be almost entirely devoid of feed residues, and the small intestine lacked normal tone and was atrophic (wasted). The liver frequently had an abnormal color that varied from a faint yellow to a deep dirty yellow. The spleen appeared to be small and atrophic and the kidneys inflamed or hemorrhagic.

Phillips and Engel (43) found lesions in the spinal cord of chicks that had received a diet deficient in pantothenic acid. The lesions were characterized by a myelin degeneration (degeneration of the sheath) of the myelinated fibers. Such degenerating fibers were found in all segments of the spinal cord down to the lumbar region. Involution (degeneration) of the thymus and liver damage also were found.

The manner in which pantothenic acid functions in the chicken is not known, but the observations of Phillips and Engel show that it is necessary for the maintenance of a normal spinal cord in the growing chick.

OCCURRENCE AND PREVENTION OF PANTOTHENIC ACID DEFICIENCY

Most of the feedstuffs ordinarily fed to poultry are fairly good sources of pantothenic acid, but diets composed largely of the cereal grains, wheat middlings, and meat scrap or fish meal may contain less of this factor than is required by the growing chick. It should also be noted that the kiln-drying of corn tends to destroy much of the pantothenic acid originally present.

Cases of dermatosis (a general name for any skin disease) have been observed among growing chicks that were being raised under practical conditions and presumably were receiving an adequate diet. Undoubtedly there are causes of dermatosis other than a deficiency of pantothenic acid, and it is possible that some of them are nutritional in nature. In the turkey, for example, a dermatosis may be produced by feeding a diet deficient in vitamin G.

The minimum pantothenic acid requirement of the chicken is set tentatively at about 5 milligrams per pound of feed. An adequate supply is about 6 milligrams per pound of feed, but apparently the diet of breeding stock should contain about 7 milligrams per pound of feed in order to insure good hatchability.

The approximate pantothenic acid contents of the richer sources of this vitamin, or vitaminlike factor, that are used in feeding poultry are:

	Milligrams per pound
Yeast, brewers', dried-----	95
Cane molasses-----	6 to 38
Peanut meal-----	25
Dried whey-----	25
Dried buttermilk-----	19
Alfalfa-leaf meal, dehydrated-----	19
Dried skim milk-----	16
Alfalfa-leaf meal-----	13
Wheat bran-----	11
Rice bran-----	11
Soybean meal-----	6

EGG-WHITE INJURY

A condition in chicks that resembles pantothenic-acid-deficiency disease may be produced by feeding diets in which all or a rather large proportion of the animal protein is derived from egg white or from whole egg. It is called egg-white injury. Even as little as 5 percent of dried egg white or an equivalent quantity of liquid egg white in such diets produces a dermatitis at the corners of the mouth and on the bottoms of the feet.

Egg-white injury does not appear if 5 to 10 times as much egg yolk as egg white is also included in the diet. It may be prevented also by including relatively large quantities of dried skim milk, or about as much dried liver as egg white, or about half as much cooked pig kidney as egg white. If the egg white is cooked before it is dried, it does not produce the dermatitis.

The first symptoms of egg-white injury is the development of a dermatitis almost simultaneously at the corners of the mouth, below the lower mandible, and on the bottoms of the feet. According to Jukes, however, roughening of the skin below the lower mandible is usually the first symptom to appear and is particularly noticeable where the skin joins the mandible. Later the eyes may be stuck shut, and fissures appear in the skin on the bottoms of the feet. If the chicks survive long enough, the fissures become numerous and rather deep.

The precise cause of egg-white injury is not known, but it appears to result from a deficiency of biotin (vitamin H—one of the group of B vitamins). Apparently the egg white, if not denatured by cooking or otherwise, combines with and inactivates the biotin unless a rather large quantity is present in the diet.

MANGANESE AND CHOLINE DEFICIENCIES*

SYMPTOMS OF PEROSIS

If young chicks are fed a diet deficient in manganese, symptoms of perosis will develop within 2 to 10 weeks, depending on the severity of the deficiency, the breed and strain of chicken, the composition of the diet, and the age at which the diet is first fed. If the deficient diet is fed from the first feeding, that is, when the chicks are 1 or 2 days old, the symptoms generally develop between the ages of 3 and 6 weeks, but if it is not fed until the chicks are 10 weeks old, the usual symptoms may not appear.

The first readily noticeable symptom is a tendency on the part of some of the chicks to rest for long periods in a squatting posi-

* This disease has been referred to at various times as hock disease, slipped tendon, and deforming leg weakness.

tion. If the tibiotarsal joints (hocks) in these chicks are carefully examined, a slight puffiness may be observed. Within a few days the joints become slightly enlarged, and frequently the skin covering them has a bluish-green cast. Apparently this is a critical stage, because in some cases, especially among the more resistant breeds or strains, the chicks frequently recover to such an extent that no readily noticeable permanent deformity results.

As the joints become further enlarged, they tend to become flattened, and the metatarsi (shank bones) and tibiae exhibit a slight bending and also often undergo a rotational twisting. As the condition continues to develop, the bones become more and more bent until gross deformity results. Frequently the articular, or joint, cartilage at the lower end of the tibia slips from its normal position, and this in turn causes the main tendons to slip from their condyles (the knucklelike ends of bones). Sometimes the curvature of a tibia is so great at its lower end that the tendons slip even though the articular cartilage has not been displaced. These changes may take place in either one or in both legs; when they take place in both legs the chicken is forced to walk on its hocks.

The symptoms of perosis in young poults and ducklings are similar to those observed in young chicks, but in the poult the next higher joint in the leg frequently becomes affected. Perosis has been found also in various wild birds, including pheasants, grouse, quail, and sparrows.

OTHER EFFECTS OF A DEFICIENCY OF MANGANESE

If adult chickens are fed a diet deficient in manganese, no observable changes in their leg joints and bones occur, but the shells of their eggs tend to become thinner and less resistant to breakage. If the deficiency is sufficiently great, egg production is decreased, and the eggs that are produced do not hatch well. The hatchability is reduced as a result of an increase in the embryonic mortality that occurs after the tenth day of incubation. According to Lyons and Insko (33) this embryonic mortality reaches its peak on the twentieth and twenty-first days of incubation, and the embryos that die after the tenth day are chondrodystrophic and characterized by very short, thickened legs, short wings, "parrot beak," a globular contour of the head, protruding abdomen, and, in the most severe cases, retarded development of the down and poor growth.

If the deficiency of manganese in the diet of laying hens is marked but not extreme, a few of the eggs may hatch. The resulting chicks may have very short leg bones, and in some cases the bones may be deformed as in chicks that develop perosis after hatching. Caskey and Norris (8) raised some of the short-legged chicks on diets that contained an adequate quantity of manganese and found that this condition of the legs persisted during a period much longer than that required for the attainment of maturity.

CHEMICAL FINDINGS AND GENERAL CONDITION OF THE BONES IN PEROSIS

The early work of Hall and King (14) indicated that chicks with slipped tendons had less bone phosphatase than normal chicks. Later Wiese, Johnson, Elvehjem, and Hart (52) observed that both the blood and bone phosphatase and the ester phosphorus of the blood are decreased in manganese deficiency in the chick.

Although the majority of workers who have studied perosis in the chicken have reported that there is no material difference in the ash content of the leg bones in perotic and normal chicks, Caskey, Gallup, and Norris (7) found that the leg bones of perotic chicks contained somewhat less ash than those of normal chicks.

Caskey, Gallup, and Norris found also that a deficiency of manganese in the diet of chicks results in a significant thickening and shortening of the bones of the legs, wings, and spinal column. In rickets, as in perosis, the leg bones may become thickened and shortened, but the shafts are poorly calcified and tough, whereas in perosis they are well calcified and relatively brittle. In osteoporosis the shafts are of normal length and much thinner than in rickets or perosis and are somewhat more springy than in perosis. In all three conditions the upper end of the tibia becomes enlarged, but it has a bulbous shape in rickets, a conical shape in perosis, and an approximately normal shape in osteoporosis.

THE COMBINED ACTION (SYNERGISM) OF MANGANESE AND CHOLINE

Soon after it was reported by Wilgus, Norris, and Heuser (54) that manganese plays an important role in preventing perosis in growing chickens, several workers observed that the addition of manganese to the diet was not in every case completely effective. In most instances no perosis occurred, but in a few instances 2 to 5 percent of the chicks developed relatively mild cases. Later it was found that the addition of manganese was less effective in preventing perosis in turkeys than in chickens.

Thus the matter stood until Jukes (25, 26) reported that choline, a widely distributed substance found in most animal and plant tissues, is effective in preventing perosis in both poults and chicks if the diet contains manganese. Workers in several institutions soon confirmed Jukes' finding that the diet must contain adequate quantities of both manganese and choline if complete protection against perosis is to be obtained.

The manner in which manganese and choline function in the development of a normal skeleton is not yet known.

OCCURRENCE AND PREVENTION OF PEROSIS

Perosis was seldom observed before the more intensive methods of raising poultry came into use. With development of out-of-season production of broilers it became a serious problem. In general, it occurred frequently whenever chickens and turkeys were raised without access to the soil. The reason is now apparent: only about 10 percent of the individual ingredients of mixed feeds for poultry contain enough manganese to furnish an adequate supply of this element, and often these feedstuffs account for only 40 percent or less of the usual feed mixtures, but when poultry have access to the soil they

ordinarily are able to obtain enough additional manganese to meet their requirement.

Corn and milk are extremely poor sources of manganese, and diets composed largely of these two ingredients are likely to cause perosis, unless additional manganese is added. On the other hand, wheat bran, wheat middlings, and rice bran are relatively rich sources, and perosis is not likely to occur on diets that contain 20 percent or more of the first two or 10 percent of the third of these feedstuffs. In any case, it is good insurance against perosis to include a small quantity of manganese sulfate in the diet.

Most research workers in poultry nutrition agree that, for adequate protection against perosis, the diet of chickens should contain about 50 parts per million of manganese. Turkeys apparently require more manganese than chickens, but 50 to 60 parts per million will ordinarily meet their requirements, provided their diet also includes a sufficient quantity of choline.

Inasmuch as most diets for poultry are likely to contain at least 20 parts per million of manganese, adequate protection against perosis generally will be obtained if about 30 parts per million are added. This quantity of manganese may be supplied easily through the use of a mixture of 100 parts, by weight, of common salt and 1.7 parts of pure anhydrous manganous sulfate, or about 2.2 parts of so-called technical grade anhydrous manganous sulfate. About 0.5 percent of this mixture should be included in all-mash diets and 1 percent in mashes with which an approximately equal quantity of grain is to be fed.

Nearly all the feedstuffs commonly used in feeding poultry contain some choline, so that typical diets for poultry are not likely to be deficient in this substance. One of the better sources of choline among feedstuffs is soybean meal, and for this reason, the inclusion of 5 to 10 percent of this feedstuff in diets for poultry, especially for turkeys, will tend to insure against a deficiency of choline.

IRON AND COPPER DEFICIENCIES AND ANEMIA

Anemia rarely if ever occurs among chickens on practical diets, but it has been demonstrated (10, 16) that it can be produced in young chicks by feeding a diet extremely deficient in iron or copper or both. The workers cited produced anemia in young chicks by feeding a diet of cow's milk, polished rice, calcium carbonate, and salt and a similar diet in which the rice was replaced by corn.

When the rice-containing diet was fed, the hemoglobin content of the blood fell from 8 grams per 100 cubic centimeters to 4 grams per 100 cubic centimeters within 12 to 15 days. The anemia was prevented by adding a small quantity of a soluble salt of iron to the diet. However, if the rice was first treated to remove the small quantity of copper it contained, it was necessary to add salts of both iron and copper to obtain normal hemoglobin formation.

When the corn-containing diet was fed, anemia developed during the early growing period but gradually disappeared as the chicks became older. As in the case of the rice-containing diet, the addition of iron prevented the occurrence of anemia.

As was found by Hogan and Parrott (22), anemia can also be produced in young chicks by feeding special, simplified diets that presumably have an adequate content of iron and copper. In such instances the anemia is the result of a deficiency of some as yet unknown nutritional factor.

Although anemia of nutritional origin occurs extremely rarely in chickens, it has been observed in developing embryos in eggs from hens that had received more or less typical diets for poultry. Anemic embryos are encountered most frequently in the fall and winter, when the parent stock does not receive much sunshine. The exact cause of this anemia in embryos is not known, but probably the cause is a deficiency of iron or copper or both. At least there is evidence that if chickens do not receive sunshine or do not have some cod-liver oil in their diet, the transfer of iron and copper to their eggs is appreciably reduced.

The available evidence on the subject indicates that under ordinary, practical conditions it is unnecessary to add compounds of iron and copper to the diets of poultry to prevent anemia. As a matter of fact, the addition of large quantities of iron compounds may produce rickets by making the phosphorus unavailable.

IODINE DEFICIENCY AND GOITER

Only a few cases of goiter, or enlarged thyroid (fig. 8), in chickens have been observed and reported in this country, but Welch (51) has stated that it is very common in Montana, and Kernkamp (28) has reported two cases in Minnesota. Goiter in the chicken

probably is more common in certain sections of this country than is generally realized. Undoubtedly the reason that only a few cases have been reported is that the enlarged thyroids are concealed by the feathers and are not readily detected. Moreover, goiter does not appear to affect the health of chickens seriously or to be a cause of heavy mortality.

Goiter has been produced experimentally in chickens by Gassner and Wilgus (13). They fed diets of extremely low iodine content (25 parts per billion) to laying hens and found enlarged thyroid glands in the chicks that hatched from their eggs.

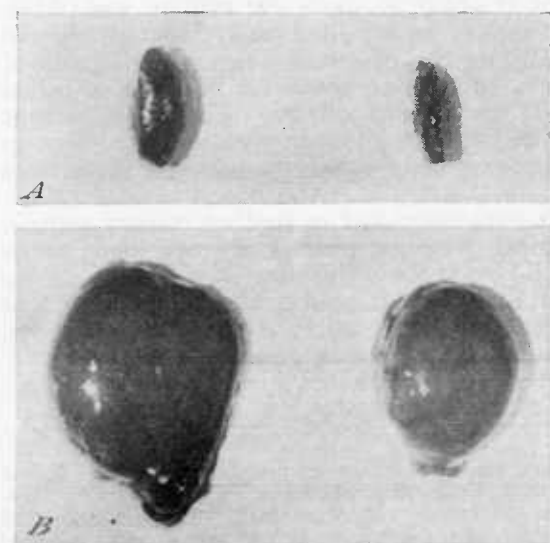


FIGURE 8.—A, normal, and B, enlarged thyroid glands of chickens. The enlarged thyroid was removed from a chicken that had been fed a diet deficient in iodine. (Courtesy of H. S. Wilgus, Colorado Agricultural Experiment Station, Fort Collins, Colo.)

Wilgus and associates (53) were able to produce goiter in young growing chicks by feeding them a simplified diet of low iodine content in which the sole protein supplement was soybeans. However, when they added sufficient iodine to this diet the thyroid glands were normal in size and structure.

Such experiments have provided definite evidence that the chicken requires a small quantity of iodine, but neither the minimum nor

the optimum amount is known. Mitchell and McClure (37) estimated that the daily iodine requirement of a 5-pound chicken is 4.5 to 9 micrograms (a microgram is one-millionth of a gram, or slightly less than one twenty-eight millionth of an ounce).

Most American workers who have studied the effect of adding small quantities of iodine to the diet of chickens have reported that no beneficial results were obtained, which suggests that typical diets for poultry are not likely to be deficient in iodine. Nevertheless, the use of so-called iodized salt in diets for poultry—especially in sections of the country where goiter is encountered in other farm animals—may be a worth-while means of insuring against a possible deficiency of iodine.

GIZZARD EROSION

Several different kinds of gizzard erosion are found in poultry, and the meager evidence now available suggests that they are the result of different causes or combinations of causes. The kind most frequently encountered is preceded by hemorrhages from the glandular layer of the gizzard, which originate from the capillaries in the submucosa. A second kind, which is less common, is characterized by a softening and a pronounced thickening of the lining of the gizzard. In a third kind, which is of still less frequent occurrence and has been observed chiefly in turkeys, the lining softens and separates completely from the glandular layer. The last kind of erosion is distinctly different from the first but resembles the second in that there is a softening of the lining.

Little or nothing is known about the development of the second and third kinds of gizzard erosion, but the histological studies of Lansing, Miller, and Titus (30) have yielded some information regarding the development of the first kind, which is discussed in the following paragraphs.

According to these authors the erosions are formed in the following way:

At one or more places in the glandular layer of the gizzard there is a seepage of blood into the secretion from which the lining is formed, and the lining is thus weakened in these places and loses some of its coherence. Reddish-brown stains in the lining, varying in size from a mere speck to several square centimeters, are evidence of such seepage. If the passage of blood into the secretion stops at this stage, the subsequent secretion yields a normal layer of lining under the affected area. After a short time threadlike, shallow fissures appear on the attrition surface of the lining in such places.

Sometimes the initial seepage of blood is followed by a pronounced hemorrhage, and blood clots form between the weakened lining and the glandular layer. If the seepage continues for some time before the hemorrhage occurs, a fairly thick but deeply stained lining may be found over the blood clot; but if the hemorrhage follows promptly after the initial seepage, only a thin lining or no lining at all is found over the site of the hemorrhage. In either case, the affected portion of the lining now lacks the backing, or support, of the glandular layer and soon cracks or sloughs off. The final result is the formation of deeply fissured areas, holes in the lining, or both.

Apparently, when the hemorrhages are large the secreting activity of the glands is markedly reduced or even stopped. In any case, new lining is not formed and large eroded areas appear.

If at any stage in the development of gizzard erosions a suitable diet is fed, the hemorrhages stop, and after 2 or 3 weeks a lining of normal appearance may be formed.

Gizzard erosion has been found in all sections of the country and presumably in all breeds of chickens. Its incidence is greatest in very young chicks and decreases with increasing age.

Apparently gizzard erosion has no appreciable effect on the rate of growth or the health of chickens. In any case, its occurrence in very young chicks is not a cause for concern. If it is found in chicks older than 4 weeks, however, the diet fed is probably not entirely satisfactory.

Various feedstuffs and other materials have been reported to be of value in clearing up gizzard erosion. Among those reputed to be of special value are dried ox bile, cholic acid, kale, hempseed meal, alfalfa products, wheat bran, wheat middlings, oats, soybean meal, pig liver and kidney, lung tissue, cartilage, and chondroitin. Of these materials the most effective are dried ox bile and cholic acid.

The diverse nature of the materials just mentioned strongly suggests that gizzard erosion may result from a deficiency of more than one nutritional factor and that the deficiency may be single or multiple. This suggestion is strengthened by the fact that although dried ox bile has been very effective in the experiments of all workers who have tested it, some workers have failed to get a response from cartilage and chondroitin, and others have obtained very little if any response from alfalfa.

FEATHER PICKING AND CANNIBALISM

Cannibalism is a term used by some poultrymen in referring to the habit sometimes developed by chickens, turkeys, other poultry, and game birds of picking one another's feathers, toes, beaks, heads, combs, backs, vents, and other parts of the body. Some poultrymen, however, restrict the use of this term to cases in which blood is drawn. Inasmuch as there are instances in which only the feathers are picked, or pulled, it seems desirable to make a distinction between feather picking and cannibalism.

Often the only result of feather picking is that some of the birds lose many of their feathers, but cannibalism nearly always leads to heavy losses through death. In flocks of pullets just starting to lay, cannibalism generally follows a case of prolapsus of the oviduct; in such cases a number of birds may become disemboweled, and rather heavy losses may result. Cannibalism among chicks often appears first in the form of toe picking, back picking, or wing picking; once established, it spreads rapidly through the flock.

Although there is evidence that feather picking and cannibalism are the result, in part, of unsatisfactory diets, there are often other contributing causes, such as overcrowding and overheating—especially in the case of chicks in battery brooders. The exact nature of the nutritional deficiency or deficiencies involved is not known, but it has been found that feather picking and cannibalism are less likely to occur if the diet contains about 20 percent of barley or oats or about 30 percent of bran and middlings.

Carver (6) has reported that feather picking and cannibalism may be controlled by using ruby-colored lights in place of ordinary lights in

battery brooders and brooder houses. Miller and Bearse (35) have reported that if oats are fed as the sole grain in diets for growing and laying pullets, cannibalism is significantly reduced. They found that the effective part of the oats was the hulls. Their findings and those of other workers indicate that feather picking and cannibalism are likely to appear if diets of very low crude-fiber content are fed.

One of the most effective methods of stopping feather picking and cannibalism is to increase the salt content of the diet for 2 or 3 days. If an all-mash diet is being fed, add 2 percent of salt, but if both mash and grain are being fed, add 4 percent of salt to the mash. Usually the feather picking or cannibalism stops within a few hours, but in some cases 2 or 3 days may be required. It should be noted that the salt treatment is recommended as a curative rather than a preventive measure; that is, it is not recommended that more than 0.5 to 0.7 percent of added salt be included regularly in all-mash diets or that more than 1 to 1.2 percent be included in mashes with which grain is fed.

Among poultrymen there is a fairly common belief that salt is poisonous to poultry, and for that reason they may be somewhat reluctant to use the salt treatment. Salt is poisonous to all animals if consumed in large single doses; Mitchell, Card, and Carman (36) have reported, however, that a daily intake of 6 to 8 grams (about 0.2 to 0.3 ounce) of salt mixed in the feed appears to have no harmful effect on chickens 9 weeks or more of age. They found that the minimum lethal dose for chickens weighing 3 to 5 pounds is equivalent to about 0.4 percent of their live weight. When the salt treatment is used, the largest quantity of salt likely to be consumed by an adult bird in a day is about 5 grams; that likely to be consumed by a growing bird is usually much less. It is thus clear that there is no danger of so-called salt poisoning when the salt treatment is used.

If the salt treatment is not effective after 2 or 3 days, it may be necessary to trim or sear back to the quick the upper mandible of the beaks of all the birds. The trimming may be done with a sharp knife, the searing with a hot soldering iron. When carefully done, the operation is painless. Ordinarily, only about three-sixteenths of an inch of the tip of the beak is removed; the proper amount can be judged readily by the appearance of the beak substance.

FLUORINE POISONING

Although the tolerance of chickens for fluorine is greater than that of cattle and swine, the continued ingestion of diets containing appreciable quantities may depress the rate of growth and the egg production. Fluorine is distributed almost universally in plants and feedstuffs, as well as in animal tissues, but the quantity present is usually very small. Danger of fluorine toxicosis, or poisoning, in chickens exists only when the drinking water contains about 2 parts per million or more of fluorine or when rock phosphate or phosphatic limestone is included in the diet. Apparently the only observable effects of fluorine toxicosis are those it has on growth and egg production.

Most of the available information about the effects of fluorine on chickens has been obtained from experiments conducted at the Wisconsin and Ohio experiment stations.

Experiments by Halpin and Lamb (15) at the Wisconsin station showed that the inclusion in the diet of chicks of 1 percent of rock phosphate that contained about 3.5 percent of fluorine had no harmful effect, but the inclusion of 2 percent depressed the growth to some extent, and the inclusion of 3 percent had a more marked effect. Only the highest of the three levels of rock phosphate in the diet of pullets tended to decrease egg production.

At the Ohio experiment station the studies of Kick, Bethke, and Record (29) showed that chicks can tolerate a larger quantity of fluorine in the form of calcium fluoride than in the form of sodium fluoride; the fluorine in rock phosphate also was more toxic than that in the calcium fluoride. They concluded that when the diet of chicks contains more than 0.036 percent of fluorine, from either sodium fluoride or rock phosphate, feed consumption and growth are decreased in proportion to the fluorine content of the diet. However, according to Hauck, Steenbock, Lowe, and Halpin (17) diets that contain as much as 0.068 percent of fluorine in the form of sodium fluoride may be fed to chicks without affecting their growth. In any case it is apparently not advisable to include rock phosphate or phosphatic limestone in diets for poultry.

SELENIUM POISONING

Practically all our knowledge of selenium toxicosis in poultry has resulted from studies conducted at the South Dakota Agricultural Experiment Station. It should be pointed out, however, that South Dakota is not the only State in which selenium toxicosis may be encountered. According to Moxon (38) selenium has been found in the soils and vegetation of at least 11 of the States in the Great Plains and the Rocky Mountains, and it is probably present in the soils of other States in these regions.

Poley, Moxon, and Franke (45) found that if laying chickens were fed diets that contained grain in which the selenium content was about 15 parts per million, feed consumption decreased appreciably, the chickens lost weight, and after about a week none of their eggs would hatch. Tully and Franke (49) observed that if chicks were fed a diet that contained 65 percent of the toxic grain their growth was definitely inhibited and their feathers became ruffled. The egg production of the pullets raised on such diets was both delayed and reduced.

In later studies Poley and Moxon (44) found that if the diet of laying chickens contained only $2\frac{1}{2}$ parts per million of selenium, hatchability was not appreciably affected; if the diet contained about 5 parts per million of selenium, however, the hatchability was reduced somewhat; and if it contained 10 parts per million the hatchability soon decreased to zero. The decrease in hatchability was attributed to abnormal development of the embryos, most of which died before the twenty-first day of incubation. The most prominent

deformity among the abnormal embryos was the lack of a full-sized upper beak. Other abnormalities were the absence of eyes, feet, and wings, wiry down, and edema of the head and neck.

OTHER DISEASES OF NUTRITIONAL ORIGIN

Several poultry-nutrition workers, while studying the effects of feeding simplified diets, have encountered various abnormal conditions in poultry that apparently could not have been caused by a deficiency of any known nutritional factor. However, the very fact that simplified diets were being fed suggests that the abnormal conditions were of nutritional origin. One such condition, the anemia described by Hogan and Parrott (22), has been mentioned in a preceding section; others are enteritis (intestinal inflammation), paralysis, arthritis, dermatosis, and fatty liver; undoubtedly there are still others.

Certain abnormal conditions, such as enteritis, dermatosis, and fatty liver have been observed even when supposedly adequate diets were being fed. As more is learned about the nutritional requirements of poultry and the nutritive properties of feedstuffs, it is probable that the causes of these abnormal conditions will be found.

ENTERITIS

Enteritis, or inflammation of the intestine—chiefly the small intestine—is frequently observed in chickens that are being raised without access to the soil and green growing plants. On autopsy, the intestine is often found to be filled with bits of shavings, straw, or other material that had been used as litter; sometimes large quantities of grit are also found. A somewhat similar but more severe condition, called ulcerative enteritis, causes heavy losses among quail, pheasants, grouse, and wild turkeys that are being raised in captivity.

Attempts to demonstrate that such conditions are caused by a micro-organism or other causative agent have failed. It has been suggested that in game birds the immediate cause is a diet of high fiber content, but enteritis is frequently encountered among both chickens and quail fed diets of comparatively low fiber content.

PARALYSIS

As has been pointed out in preceding sections, a deficiency of vitamin E in the diet of the young growing chicken produces lesions in the brain, of pantothenic acid in the spinal cord, of vitamin G in the main peripheral nerve trunks, of vitamin A in the central and peripheral nervous systems. Moreover, a dietary deficiency of vitamin B₁ produces a toxicosis, or poisoning, of the nervous system. Accordingly, a deficiency of one or more of these vitamins may produce paralysis or a similar condition.

Paralysis of nutritional origin has been observed, however, when adequate quantities of all five of the vitamins just mentioned were

supplied. For example, Jukes and Babcock (27) have described a paralysis that could be prevented by supplementing the diet with alfalfa meal or a water extract of alfalfa, and Bird and Oleson (4) have described a condition, in which there is incoordination of the leg muscles, which they attribute to a deficiency of vitamin B₄. According to Hegsted, Oleson, Elvehjem, and Hart (19) the latter condition is not prevented by alfalfa or vitamin E but is prevented by relatively large quantities of dried brain, cartilage, wheat middlings, yellow corn, or wheat.

ARTHRITIS

In 1935 Van der Hoorn, Branion, and Graham (50) described a deformity of the legs of chickens that resulted from feeding simplified diets that contained highly purified casein. They tentatively called the condition arthritis. Later Branion and his associates (5) concluded that this condition is probably the result of a deficiency of one or more inorganic elements. Still later other workers (19) suggested that the "paralysis" (see the preceding discussion of paralysis) that they had encountered in the chicken might be the same as the "arthritis" reported by Van der Hoorn, Branion, and Graham (50).

According to the latter authors, the first symptom of this arthritis appeared when the chicks were about 3 weeks old. At first the chicks were merely less active than usual, but within a few days they showed very little inclination to walk, and when they did walk their gait was decidedly stilted, and there was practically no flexion of the tibiotarsal (hock) joints. At this stage the capsule of the joint was swollen and somewhat congested, and there was a slight excess of fluid in the cavity. Gradually the symptoms became more pronounced until the chicks refused to walk. Often the leg bones became deformed as in perosis, but the investigators concluded that their "arthritis" was not perosis.

DERMATOSIS

From time to time a dermatosis similar to that produced by a deficiency of pantothenic acid or to that which results from the feeding of egg white is observed among growing chickens that are receiving supposedly adequate diets. This condition often disappears if a complete change of diet is made, but it is not cured by adding rich sources of pantothenic acid to the original diet.

Hegsted and associates (18) have reported such a condition occurring among chicks that were fed a purified diet in which there was an adequate supply of pantothenic acid. In many respects the symptoms were the same as those of egg-white injury, and complete cures were obtained by injecting a potent preparation of vitamin H (biotin). Hegsted and associates concluded that it is possible that all proteins have the effect of egg white to some extent but that the effect would be evidence only when purified diets low in the protective factor are fed.

FATTY LIVER

When growing chickens are fed certain simplified diets, their livers often have an abnormal yellow color and show evidence of fatty degeneration. Lepkovsky, Taylor, Jukes, and Almquist (32) have reported that a deficiency of vitamin G (riboflavin) causes fatty liver in chicks, and Engel and Phillips (12) have reported

that when vitamin B₁ is administered to chicks that have been on a diet deficient in this vitamin a similar condition develops. However, Hegsted, Oleson, Elvehjem, and Hart (19) have described a fatty degeneration of the liver that could not be attributed to either of the causes just mentioned.

The available evidence indicates that fatty liver in chickens may be the result of a number of causes and not a single, specific nutritional deficiency. When fatty liver is found, however, it may be concluded that the diet is unsatisfactory, as a result either of one or more deficiencies or of an imbalance of certain nutritional factors.

EFFECT OF NUTRITIONAL DEFICIENCIES ON GROWTH AND REPRODUCTION

Although an animal's capacity to grow is an inherited character, the growth it makes depends on its nutrition. The dependence of



FIGURE 9.—Many nutritional deficiencies retard growth. Both these chickens are 114 days old. The one at the left received an ordinary mixed diet; the one at the right received a diet deficient in vitamin C. (Courtesy of Thomas H. Jukes, Division of Poultry Husbandry, California Agricultural Experiment Station, Davis, Calif.)

growth on nutrition is so great that when an adequate diet is fed the relationship between live weight and feed consumption may be expressed with a high degree of accuracy by a mathematical equation. When an inadequate diet is fed, the animal's growth is

usually retarded and irregular. Often a retardation of growth is the first indication that the diet is deficient.

A fairly large number of nutritive factors are required for normal growth. Among those that play especially prominent roles in maintaining growth in poultry are vitamins A, B₁, B₆, D, and G (fig. 9), pantothenic acid, glucuronic acid, choline, several of the amino acids, and many of the inorganic elements. Apparently, there are other vitamins or vitaminlike factors that affect growth, but very little is yet known about them.

Obviously, a retardation of growth merely indicates that the diet is inadequate. Only when other symptoms appear, or when information about the diet that is being fed is fairly complete, is it possible to identify the deficiency. The symptoms of a number of nutritional deficiencies have already been described.

Egg production sometimes continues even though a deficient diet is fed. Likewise the fertility of the eggs appears not to be greatly affected by dietary deficiencies unless they are acute and prolonged enough to affect the health of the birds, especially that of the males. The hatchability of the eggs is readily decreased by a number of dietary deficiencies. However, as a matter of fact, the first and frequently the only indication that the diet of adult birds is deficient in one or more nutritive factors is a low hatchability of the eggs.

Among the nutritional factors known to be required for the production of hatchable eggs are vitamins A, D, E, and G, pantothenic acid, protein of good quality, calcium, and manganese. To this list may be added the "alcohol-precipitate factor" of Schumacher and Heuser (47). Undoubtedly, other factors are required, and as more work is done they will be discovered and described.

Nutritional deficiencies are not the only causes of poor hatchability. As was mentioned in the discussion of selenium poisoning, if the diet of the dams contains as much as 10 parts per million of selenium none of their eggs will hatch. Moreover, the inclusion of excessively large quantities of calcium or phosphorus in the diet also decreases hatchability.

Just how a deficiency of vitamin A in the diet of the dams affects the development of the embryos in their eggs is not known, but the ultimate effect—a decreased hatchability—may be easily demonstrated.

When the diet of the dams is deficient in vitamin D, the embryos in their eggs are unable to obtain enough calcium and phosphorus, and the embryonic mortality reaches a peak on the eighteenth or nineteenth day of incubation. An excessive intake of vitamin D (5 or 6 times the normal requirement) also decreases the hatchability of the eggs.

A vitamin E deficiency in the diet of the dams is manifested by a marked increase in the embryonic death rate between the third and fifth days of incubation.

When there is a deficiency of vitamin G or of protein of good quality, a marked increase in the so-called second-week embryonic mortality occurs.

A deficiency of pantothenic acid in the diet reduces the hatcha-

bility of the eggs, but no characteristic peak of embryonic mortality has been reported.

A deficiency of calcium in the diet seems to have an effect similar to that of a deficiency of vitamin D. An excess of either calcium or phosphorus causes an increase in embryonic mortality during the last 3 days of the incubation period.

The effect on the embryos of feeding diets deficient in manganese to chickens has been previously discussed.

LITERATURE CITED

- (1) ALMQUIST, H. J., and STOKSTAD, E. L. R.
1935. HEMORRHAGIC CHICK DISEASE OF DIETARY ORIGIN. *Jour. Biol. Chem.* 111: 105-113.
- (2) BARGER, EDGAR HUGH, and CARD, LESLIE ELLSWORTH.
1938. DISEASES AND PARASITES OF POULTRY. Ed. 2, 386 pp., illus. Philadelphia.
- (3) BIRD, H. R., and CULTON, THOS. G.
1940. GENERALIZED EDEMA IN CHICKS PREVENTED BY D. L-ALPHA TOCOPHEROL. *Soc. Expt. Biol. and Med. Proc.* 44 (2): 543-547, illus.
- (4) ——— and OLESON, J. J.
1938. VITAMIN A DEFICIENCY IN CHICKS FED PURIFIED RATIONS CONTAINING COD LIVER OIL. *Soc. Expt. Biol. and Med. Proc.* 38: 870-871.
- (5) BRANION, H. D., MARTIN, R. L., ROBERTSON, E. B., and others.
1938. THE VARIATION IN THE NUTRITIVE VALUE OF CASEIN. *Poultry Sci.* 17: 301-316.
- (6) CARVER, J. S.
1931. THE CONTROL OF CANNIBALISM IN BATTERY BROODERS AND FATTENING BATTERIES. *Poultry Sci.* 10: 275-277.
- (7) CASKEY, C. D., GALLUP, W. D., and NORRIS, L. C.
1939. THE NEED FOR MANGANESE IN THE BONE DEVELOPMENT OF THE CHICK. *Jour. Nutr.* 17: 407-417, illus.
- (8) ——— and NORRIS, L. C.
1940. MICROMELIA IN ADULT FOWL CAUSED BY MANGANESE DEFICIENCY DURING EMBRYONIC DEVELOPMENT. *Soc. Expt. Biol. and Med. Proc.* 44: 332-335, illus.
- (9) DAM, HENRIK, and GLAVIND, JOHANNES.
1940. VITAMIN E UND KAPILLARPERMEABILITÄT. *Naturwissenschaften* 28: 207.
- (10) ELVEHJEM, C. A., and HART, E. B.
1929. THE RELATION OF IRON AND COPPER TO HEMOGLOBIN SYNTHESIS IN THE CHICK. *Jour. Biol. Chem.* 84: 131-141.
- (11) ENGEL, R. W., and PHILLIPS, P. H.
1938. THE LACK OF NERVE DEGENERATION IN UNCOMPLICATED VITAMIN B₁ DEFICIENCY IN THE CHICK AND THE RAT. *Jour. Nutr.* 16: 585-596.
- (12) ——— and PHILLIPS, P. H.
1939. FATTY LIVERS AS A RESULT OF THIAMIN ADMINISTRATION IN VITAMIN B₁ DEFICIENCY OF THE RAT AND THE CHICK. *Jour. Nutr.* 18 (4): 329-338, illus.
- (13) GASSNER, F. X., and WILGUS, H. S.
1940. CONGENITAL GOITER IN CHICKS. (Abstract of paper) *Poultry Sci.* 19: 349.
- (14) HALL, G. E., and KING, EARL J.
1931. CALCIUM AND PHOSPHORUS METABOLISM IN THE CHICKEN. II. "RANGE PARALYSIS." *Poultry Sci.* 10: 259-268, illus.
- (15) HALPIN, J. G., and LAMB, ALVIN R.
1932. THE EFFECT OF GROUND PHOSPHATE ROCK FED AT VARIOUS LEVELS ON THE GROWTH OF CHICKS AND ON EGG PRODUCTION. *Poultry Sci.* 11: 5-13.
- (16) HART, E. B., ELVEHJEM, C. A., KEMMERER, A. R., and HALPIN, J. G.
1930. DOES THE PRACTICAL CHICK RATION NEED IRON AND COPPER ADDITIONS TO INSURE NORMAL HEMOGLOBIN BUILDING? *Poultry Sci.* 9: 92-101.

- (17) HAUCK, HAZEL M., STEENBOCK, H., LOWE, JAMES T., and HALPIN, J. G.
1933. EFFECT OF FLUORINE ON GROWTH, CALCIFICATION AND PARATHYROIDIS IN THE CHICKEN. *Poultry Sci.* 12: 242-249, illus.
- (18) HEGSTED, D. MARK, OLESON, J. J., MILLS, R. C., and others.
1940. STUDIES ON A DERMATITIS IN CHICKS DISTINCT FROM PANTOTHENIC ACID DEFICIENCY. *Jour. Nutr.* 20: 599-606, illus.
- (19) ——— OLESON, J. J., ELVEHJEM, C. A., and HART, E. B.
1940. THE ESSENTIAL NATURE OF A NEW GROWTH FACTOR AND VITAMIN B₆ FOR CHICKS. *Poultry Sci.* 19: 167-176.
- (20) HEYWANG, BURT W., and MORGAN, RUDOLPH B.
1937. OBSERVATIONS ON SOME SYMPTOMS OF VITAMIN A DEFICIENCY IN CHICKS. *Poultry Sci.* 16: 388-392, illus.
- (21) HINSHAW, W. R., and LLOYD, W. E.
1934. VITAMIN-A DEFICIENCY IN TURKEYS. *Hilgardia* 8: 281-304, illus.
- (22) HOGAN, ALBERT G., and PARROTT, ERNEST M.
1940. ANEMIA IN CHICKS CAUSED BY A VITAMIN DEFICIENCY. *Jour. Biol. Chem.* 132: 507-517, illus.
- (23) JUKES, THOMAS H.
1939. VITAMIN B₆ DEFICIENCY IN CHICKS. *Soc. Expt. Biol. and Med. Proc.* 42: 180-182, illus.
- (24) ———
1940. EFFECT OF YEAST EXTRACT AND OTHER SUPPLEMENTS ON THE GROWTH OF CHICKS FED SIMPLIFIED DIETS. *Jour. Biol. Chem.* 133: 631-632.
- (25) ———
1940. PREVENTION OF PEROSIS BY CHOLINE. *Jour. Biol. Chem.* 134: 789-790.
- (26) ———
1940. EFFECT OF CHOLINE AND OTHER SUPPLEMENTS ON PEROSIS. *Jour. Nutr.* 20: 445-458, illus.
- (27) ——— and BABCOCK, SIDNEY H., JR.
1938. EXPERIMENTS WITH A FACTOR PROMOTING GROWTH AND PREVENTING PARALYSIS IN CHICKS ON A SIMPLIFIED DIET. *Jour. Biol. Chem.* 125: 169-181, illus.
- (28) KERKAMP, H. C. H.
1925. GOITER IN POULTRY. *Amer. Vet. Med. Assoc. Jour.* 67: 223-228, illus.
- (29) KICK, C. H., BETHKE, R. M., and RECORD, P. R.
1933. EFFECT OF FLUORINE ON THE NUTRITION OF THE CHICK. *Poultry Sci.* 12: 382-387.
- (30) LANSING, ALBERT I., MILLER, DAVID, and TITUS, HARRY W.
1939. THE FORMATION OF EROSIONS OF THE GIZZARD LINING IN THE YOUNG CHICK. *Poultry Sci.* 18: 475-480, illus.
- (31) LEPKOVSKY, SAMUEL, and JUKES, THOMAS H.
1936. THE RESPONSE OF RATS, CHICKS, AND TURKEY POULTS TO CRYSTALLINE VITAMIN G (FLAVIN). *Jour. Nutr.* 12: 515-526, illus.
- (32) ——— TAYLOR, L. W., JUKES, T. H., and ALMQUIST, H. J.
1938. THE EFFECT OF RIBOFLAVIN AND THE FILTRATE FACTOR ON EGG PRODUCTION AND HATCHABILITY. *Hilgardia* 11: 559-591, illus.
- (33) LYONS, MALCOLM, and INSKO, W. M., JR.
1937. CHONDRODYSPLASIA IN THE CHICK EMBRYO PRODUCED BY MANGANESE DEFICIENCY IN THE DIET OF THE HEN. *Ky. Agr. Expt. Sta. Bul.* 371: 61-75, illus.
- (34) MCGOWAN, JOHN POOL, and EMSLIE, ARTHUR RAYMOND GORDON.
1934. RICKETS IN CHICKENS, WITH SPECIAL REFERENCE TO ITS NATURE AND PATHOGENESIS. *Biochem. Jour.* 28: [1503]-1512, illus.
- (35) MILLER, M. WAYNE, and BEARSE, GORDON E.
1937. THE CANNIBALISM PREVENTING PROPERTIES OF OATS. *Poultry Sci.* 16: 314-321.
- (36) MITCHELL, H. H., CARD, L. E., and CARMAN, G. G.
1926. THE TOXICITY OF SALT FOR CHICKENS. *Ill. Agr. Expt. Sta. Bul.* 279: 133-156, illus.
- (37) ——— and McCURE, F. J.
1937. MINERAL NUTRITION OF FARM ANIMALS. *Natl. Res. Council Bul.* 99, 135 pp.

- (38) MOXON, ALVIN L.
1937. ALKALI DISEASE OR SELENIUM POISONING. S. Dak. Agr. Expt. Sta. Bul. 311, 91 pp., illus.
- (39) NITZESCU, I. I., and IOANID, V.
1940. LA GLYCÉMIE CHEZ LES POULES EN AVITAMINOSE B. Soc. de Biol. [Paris] Compt. Rend. 133: 490-491, illus.
- (40) NORRIS, L. C., WILGUS, H. S., JR., RINGROSE, A. T., and others.
1936. THE VITAMIN-G REQUIREMENT OF POULTRY. N. Y. (Cornell) Agr. Expt. Sta. Bul. 660, 20 pp., illus.
- (41) PAPPENHEIMER, ALWIN M., GOETTSCH, MARIANNE, and JUNGHER, ERWIN.
1939. NUTRITIONAL ENCEPHALOMALACIA IN CHICKS AND CERTAIN RELATED DISORDERS OF DOMESTIC BIRDS. Conn. (Storrs) Expt. Sta. Bul. 229, 121 pp., illus.
- (42) PHILLIPS, PAUL H., and ENGEL, R. W.
1938. THE HISTOPATHOLOGY OF NEUROMALACIA AND "CURLED TOE" PARALYSIS IN THE CHICK FED LOW RIBOFLAVIN DIETS. Jour. Nutr. 16: 451-463, illus.
- (43) ——— and ENGEL, R. W.
1939. SOME HISTOPATHOLOGIC OBSERVATIONS ON CHICKS DEFICIENT IN THE CHICK ANTIDERMATITIS FACTOR OR PANTOTHENIC ACID. Jour. Nutr. 18: 227-232, illus.
- (44) POLEY, W. E., and MOXON, A. L.
1938. TOLERANCE LEVELS OF SELENIFEROUS GRAINS IN LAYING RATIONS. Poultry Sci. 17: 72-76, illus.
- (45) ——— MOXON, A. L., and FRANKE, K. W.
1937. FURTHER STUDIES OF THE EFFECTS OF SELENIUM POISONING ON HATCHABILITY. Poultry Sci. 16: 219-225, illus.
- (46) RINGROSE, A. T., NORRIS, L. C., and HEUSER, G. F.
1931. THE OCCURRENCE OF A PELLAGRA-LIKE SYNDROME IN CHICKS. Poultry Sci. 10: 166-177, illus.
- (47) SCHUMACHER, A. E., and HEUSER, G. F.
1940. SOME PROPERTIES OF THE ALCOHOL-PRECIPIRATE FACTOR WITH FURTHER RESULTS OF ITS EFFECTS ON CHICKS AND HENS. Poultry Sci. 19: 315-320.
- (48) STOKSTAD, E. L. R., and MANNING, P. D. V.
1938. THE EFFECT OF RIBOFLAVIN ON THE INCIDENCE OF CURLED TOE PARALYSIS IN CHICKS. Jour. Nutr. 16: 279-283.
- (49) TULLY, W. C., and FRANKE, K. W.
1935. A NEW TOXICANT OCCURRING NATURALLY IN CERTAIN SAMPLES OF PLANT FOODSTUFFS. VI. A STUDY OF THE EFFECT OF AFFECTED GRAINS ON GROWING CHICKS. Poultry Sci. 14: 280-284, illus.
- (50) VAN DER HOORN, R., BRANION, H. D., and GRAHAM, W. R., JR.
1935. STUDIES IN THE NUTRITION OF THE CHICK. II. EFFECT OF PURIFICATION OF CASEIN IN SIMPLIFIED DIET. Poultry Sci. 14: 285-290, illus.
- (51) WELCH, HOWARD.
1928. GOITER IN FARM ANIMALS. Mont. Agr. Expt. Sta. Bul. 214, 26 pp., illus.
- (52) WIESE, A. C., JOHNSON, B. C., ELVEHJEM, C. A., and HART, E. B.
1938. PHOSPHORUS METABOLISM OF CHICKS AFFLICTED WITH PEROSIS. Science 88: 383-384.
- (53) WILGUS, H. S., GASSNER, F. X., PATTON, A. R., and GUSTAVSON, R. G.
1940. THE GOITROGENICITY OF SOYBEANS. (Abstract of paper) Poultry Sci. 19: 366.
- (54) WILGUS, H. S., JR., NORRIS, L. C., and HEUSER, G. F.
1936. THE RÔLE OF CERTAIN INORGANIC ELEMENTS IN THE CAUSE AND PREVENTION OF PEROSIS. Science 84: 252-253.

At the time this book went to press, the drugs and other materials mentioned in various articles—chiefly as disinfectants, insecticides, and anthelmintics—were still available for veterinary and medical use. Under war conditions, however, it is possible that some of these materials may become scarce or unavailable. In that case, the reader should obtain professional advice from the Department of Agriculture, the State experiment station, a local veterinarian, or the county agent as to available substitutes.